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Mutual influence of lipid-antioxidant-surfactant in microheterogeneous systems

O.T. Kasaikina

N. N. Semenov Institute of Chemical Physics, Russian Academy of Sciences,4 Kosygin Str., 119991 Moscow, Russian Federation

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This short review focuses specifically on the current understanding of the role of microheterogeneous reaction medium and surfactants in particular, in preventing and inhibiting lipid oxidation, and on the influence of surfactants on the behavior of known natural and synthetic antioxidants (AO). The mutual influence of the components and the conditions of occurrence of synergism and antagonism in a complex system (lipid – antioxidant – surfactant) are discussed as well.

Key words: Free radicals, Hydroperoxides, Antioxidants, Surfactants, Synergism, Antagonism

INTRODUCTION

After Millennium, there is a steady trend in the use of natural products in innovative technologies for the creation of healthy and healing foods, cosmetics, and medicines containing essential unsaturated lipids and natural antioxidants (AO) because of consumer demand for natural ingredients [1-3]. Every drugstore sells a large set of individual antioxidants and their mixtures in the free market. Antioxidants are classified into two broad divisions, depending on whether they are soluble in water (hydrophilic) or in lipids (lipophilic). Multicomponent microheterogeneous systems usually contain surface-active substances (surfactants, S), which may have an impact not only on the structure and on the distribution of components in the system, but also on the reactivity and chemical behavior of key components. It must be noted that living organisms as a whole and individual cells can be considered as an open biochemical reactor of full displacement with multicomponent microheterogeneous medium as well. In recent years, a number of studies have produced evidence that for the development of innovative antioxidant technologies that prolong the quality of lipid containing products, for properly using of antioxidant drugs, multiphase and boundary effects in microheterogeneous systems containing surfactant, and mutual influence of lipids-antioxidants - surfactants have to be taken into account [4-8]. This short review focuses specifically on the current understanding of the role of microheterogeneous reaction medium and surfactants in particular, in preventing and inhibiting lipid oxidation, and the influence of surfactants on the behavior of known natural and synthetic antioxidants (AO). The mutual influence of the components and the conditions of occurrence of synergism and antagonism in a complex system of (lipid–antioxidant–surfactant) are discussed as well.

Pro- and antioxidant effect of surfactant on lipid and hydrocarbon oxidation

For a long time (all the last century) hydrocarbon and lipid (LH) oxidation by oxygen was considered as a free radical chain branching process [8-13]:

$$\mathrm{LH} \xrightarrow{O_2,\ L^{\bullet},\ LO_2\bullet} \mathrm{LOOH} \xrightarrow{} \begin{matrix}\text{Alcohols}\\ \text{Ketones}\\ \text{Aldehydes}\\ \text{Acids…}\end{matrix}$$

$$\mathrm{LOOH} \rightarrow LO^{\bullet}, LO_2^{\bullet}$$

Scheme 1.

The rate of the chain process (W_{O2}) is the product of the initiation rate (W_i) on the chain length (ν):

$$W_{O2} = W_i \cdot \nu \qquad (1)$$

Hydroperoxides (LOOH) – the primary oxidation products play the key role in the radical initiation. The chain initiation rate can be described by eqn. (2):

$$W_i = W_0 + e\ k_d\ [LOOH] \qquad (2)$$

where, W_0 is the initiation rate without LOOH participation, k_d – apparent rate constant and e – the so called „radical escape“ during LOOH decomposition. So, the additives which affect the LOOH decomposition can regulate the whole oxidation rate. For example, transient metals, which catalyze LOOH decomposition into free radicals, accelerate oxidation. The additives, which reduce

* To whom all correspondence should be sent:
E-mail: : okasai@yandex.ru

LOOH into molecular products without radical formation, decrease oxidation rate.

In multicomponent microheterogeneous systems such significant factors as the uneven distribution of reagents within the scope and the influence of environment polarity on their reactivity, can greatly affect the oxidation rate and the product composition [4-7,15-17]. It is important that hydroperoxides and surfactants (S) form mixed micelles in microheterogeneous systems, direct micelles in water solutions (O/W) and reverse ones (W/O) in lipid medium:

$$mLOOH + nS \Leftrightarrow (mLOOH\ldots nS) \quad (3)$$

The formation of mixed micelles was studied and confirmed by different methods such as tensiometry, NMR, DLS [15,17,18]. The charge of surfactants is currently considered as one of the main factors in the oxidative stability of O/W systems because it governs attractive or repulsive forces (the Coulomb interaction of charges) between metal ion in water and interfaces [6,19,20], where LOOH and other amphiphilic compounds are concentrated. By this reason, O/W lipid systems stabilized by cationic surfactants (S^+) were found to be more oxidative stable than systems stabilized with anionic (S^-) surfactants [19,21].

In the reverse systems W/O, the influence of surfactant's charges is weakened in oil medium and reverse dependence on the nature of the surfactant is observed. Cationic surfactants (S^+) were found to be pro-oxidants because they accelerate the oxidation of hydrocarbons and lipids [17,18, 22-24]. The key reaction, which causes the acceleration, is the catalytic decomposition of LOOH into free radicals in mixed micelles ($mLOOH...nS^+$) [17,18,22]. In these micelles, the peroxide bond is evidently located in a strong electrical field of the electrical double layer with strength of ~10^5—10^7 $V \cdot m^{-1}$, weakening bond –O-O- and stimulating the homolytic decomposition of LOOH. The activation energy of the LOOH thermal decomposition is 90—120 kJ mol^{-1}, whereas in micelles with cationic surfactants the activation energy decreases to 40—60 kJ mol^{-1}. The polar metal compounds concentrate in the reversed micelles and further accelerate the homolysis of LOOH with the formation of radicals. Synergism between cationic surfactants and transient metals was observed in accelerated oxidation of ethylbenzene and limonene [23, 24].

The influence of anionic surfactants (S^-) in the systems W/O strongly depends on the hydroperoxide nature and on the structure of the polar head of the surfactant. Bright mutual influence of "lipid - surfactant" resulted in strong antioxidant effect that was discovered in the system "alkylaromatic hydrocarbons – sodium dodecyl sulfate (SDS) or alkylphosphates". SDS completely suppresses the oxidation of ethylbenzene and other alkylaromatic hydrocarbons [17, 25], but it does not affect unsaturated lipid oxidation [25]. The decomposition of ethylbenzene and cumene hydroperoxides in the presence of SDS occurs without free radical generation and results in the formation of phenol and corresponding carbonyl compound. Very low rate of radical initiation (only *via* chain origin in thermal reactions of O_2 and LOOH) and the resulting phenol together provide effective inhibition of the oxidation of alkylaromatic hydrocarbons. It is shown in [26] that synergism of the inhibiting action of surfactants SDS and trialkyl phosphates and alkylaromatic hydrocarbons is observed in the mixtures of decane and about 10% of alkylaromatic ethybenzene and cumene.

Nonionic and zwitter-ionic surfactants and some proteins have been generally found to better protect the lipid phase against oxidation in both O/W [20, 26-28] and W/O systems [26,29,30] due to their ability to form thick layers at the interface and to separate the hydrophilic initiator and the lipid substrate. Popular synthetic nonionic surfactants Triton X-100, Tweens and Pluronics along with zwitter-ionic lecithins are widely used in food and medical industry because they are non-toxic and non-expensive. They form direct micelles in water solution. Because they include a hydrophilic polyethylene oxide chain, these surfactants can be oxidized by a chain mechanism in the presence of radical initiators [28]. Under equal conditions, the surfactant activity in the chain radical oxidation decreases in the order: PC > TX-100 > F-68 >Tween-65 [28]. Here PC is egg phosphatidylcholine. A less reactive TX-100 acts as an "antioxidant" in the oxidation of phosphatidylcholine. In the case of a mixture of PC and TX-100, the rate of oxidation is less than the rates of individual components. Measurements of micellar sizes showed that TX-100 coats multilamellar liposomes of PC and thus protects PC from oxidation initiated in water phase. Fig. 1 shows the possibility of egg phosphatidylcholine (PC) to inhibit lipid oxidation in the model experiment of limonene oxidation, catalyzed by hydrophilic colloidal catalyst on the base of Fe(III) oxide [30,31]. This catalyst facilitates limonene hydroperoxide decomposition into free radicals and thereby it accelerates limonene oxidation which occurs *via* free radical chain mechanism and can be stopped by a chain- breaking inhibitor (Fig. 1, curve 1).

The addition of PC results in the decrease of O_2 uptake rate. However, the double addition of the same inhibitor does not affect the oxidation rate. It

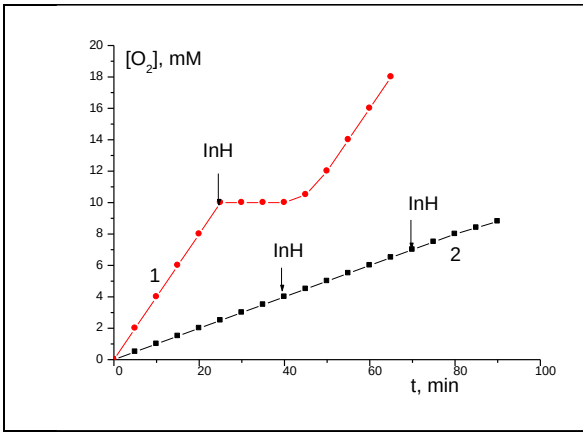


Figure 1. Kinetic curves of O_2 uptake during catalytic oxidation of (*1*) limonene and (*2*) limonene containing lecithin (PC) at the following compositions of reaction mixtures. (*1*) 4 mL of 1 M limonene solution in chlorobenzene and 32 mM LOOH + 21 μL catalyst suspension; (*2*) the same + 0.132 g lecithin; 60°C.The overall concentration of Fe^{3+} in both experiments is 1×10^{-4} M. The moments of addition of the inhibitor InH (0.023 mM) are shown by arrows.

means that PC solubilizes all the hydrophilic colloid catalyst which affects only PC capsule oxidation and the escape of radicals into bulk limonene is hampered. The similar shielding effect of PC on the escape of radicals generated by a hydrophilic micellar system (acetylcholine – hydroperoxide) in n-decane solution is described in [29].

The synergism of PC with α-tocopherol (vitamin E) in inhibiting the peroxidation of edible lipids was observed and investigated by many authors in various lipids and model systems [33-36]. It must be noted that the mechanism of synergism for phospholipids and α-tocopherol includes a colloid protective factor as well. The reactions of chain transfer by radicals derived from the inhibitor are known as a kinetic property of unhindered phenoxyl radicals, tocopheryl-radicals, in particular [9,13]. The chain transfer reactions to a marked degree decrease the antioxidant efficacy [37]. Cooperation of amphiphilic tocopherols and tocopheryl-radicals with PC can hamper the chain transfer and increase oxidative stability of oil. May be by this reason, lipids extracted from Antarctic Crill containing high content of PC and α-tocopherol demonstrated the highest antioxidant activity in β-carotene oxidation as compared with sunflower oil, lard and individual α-tocopherol taken in the equal quantity under the same conditions [26].

We have considered a few mutual synergistic effects in the system "S-LH-AO" resulted in more pronounced inhibition of oxidation. But there are antagonistic effects in lipid-antioxidant-surfactant mixtures which have to be detected and taken into account. First of all, antagonism of cationic surfactants and AO in the lipid W/O systems. The key reaction is the accelerated decomposition of LOOH into free radicals in the mixed micelles (mLOOH…nS^+). In inhibited oxidation, pro-oxidant action of cationic surfactants may increase due to direct oxidation of amphiphilic AO by a hydroperoxide, activated in the mixed micelles [38]. The mixture of PC with $CaCl_2$ appeared to be antagonistic to β-carotene in limonene oxidation. Calcium is not a transient metal, and Ca^{2+} does not affect LOOH decay. However, the mixture PC + $CaCl_2$ accelerates β-carotene consumption because of the release of choline from the zwitter-ionic polar head of PC and transformation of PC into a cationic surfactant, which catalyzes LOOH decay into free radicals [39].

Mutual influence of (thiol-lipid- other AO) on oxidative stability

Special attention has to be paid to the peculiar pro- and antioxidant properties of thiols such as important endogenous glutathione, cysteine and homocysteine, as well as a number of SH-containing substances used in food, cosmetics and drug production.

Thiols (RSH) are known as preventive antioxidants reducing hydroperoxides and H_2O_2 into molecular products [8-13, 40]. Natural thiols cysteine and especially glutathione (GSH) which concentration in living cells is rather high (several mM) are of great importance for living organisms and are considered as bioantioxidants. However, thiols are a potential source of thiyl radicals, which are known to catalyze *cis-trans*- isomerization of double bonds [41-43]. Molecules of unsaturated fatty acid present in the living organisms and high-quality natural oils adopt the *cis*-configuration. *Trans*-isomers appear in the course of hydrogenation and high-temperature treatment of natural fats and oils. In living organisms, *trans*-lipids incorporate into cell membranes and thus violate the balance of exchange processes [44]. The rate of c*is—trans*-isomerization caused by thiyl radicals decreases in the presence of oxygen, and phenolic antioxidants inhibit isomerization through chain termination [45]. In the last two decades, along with *cis-trans* isomerization, the thiol-ene reactions attract much attention as means of synthesis of hetero-chain-compounds. These reactions occur *via* radical-chain mechanism and are accelerated by light and initiators [46-49]. Recently [50], the simplest thiol

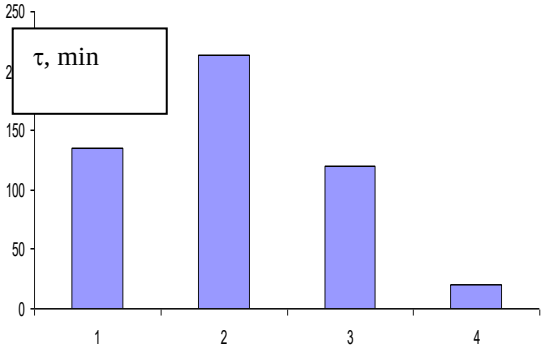
Table 1. Rates of O_2 uptake (W_{O2}) and results of the analysis of the content of trans-isomers in 1 hour of oxidation of methyl linoleate (0.2M), initiated by AIBN (5mM) at 50°C in n-decane solution in the presence of mercaptoethanol, RSH, (50mM) and diphenylamine, DA (5mM) and in parallel experiments conducted under nitrogen atmosphere.

No	System	W_{O2} 10^9, mol·(L·s)$^{-1}$	Percentage of *trans*-isomers in 1 h under O_2, %	Percentage of *trans*-isomers in 1 h under N_2, %
1	ML	6.1	0	0
2	ML+DA	2.1	0.2	0.3
3	ML+RSH	7.7	7.1	14.2
4	ML+DA+RSH	9,7	11.5	15.8

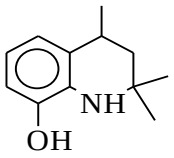
mercaptoethanol (RSH) was found to accelerate the oxidation of hydrocarbons and methyl linoleate due to the interaction of RSH with hydroperoxides resulting in a low yield of free radicals. In combination with phenolic AO, mercatoethanol shows synergism of the antioxidant action, whereas with the aromatic amines RSH exhibits antagonism.

The data of Table 1 demonstrate the antagonistic effect of mercaptoethanol additives (50 mM) on the rate of methyl linoleate (ML) oxidation, initiated by 5 mM of AIBN (azobisisobutyronitrile) and inhibited by diphenylamine (5mM). It is seen that RSH alone gently accelerates O_2 uptake and stimulates *cis-trans*-isomerization, which decelerates under oxygen atmosphere. DA added alone inhibits the oxidation. However, when DA and RSH are added together, it results in the increase of both the rates of ML oxidation and *cis-trans*-isomerization.

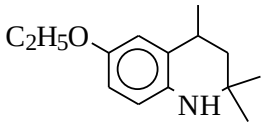
The differences in the behavior of RSH towards phenolic AO and aromatic amines can be brightly illustrated by the comparison of the induction periods caused by a mixture of RSH with two hydrogenated quinolines HQ1 and HQ2 (Figure 2). These compounds are chain-breaking AO of great efficacy [51,52]. The rate constants for the reaction of HQ1 and HQ2 with peroxyl radicals are higher than 10^6 L(mol s)$^{-1}$.



(a)



HQ1 (b)



HQ2 (c)

Figure 2. (a) Induction periods in the oxidation of methyl linoleate ([ML] = 0.2 M, [AIBN] = 4 mM) in decane at 50°C in the presence of $3 \cdot 10^{-5}$M hydrogenated quinolines HQ1 (1,2) and HQ2 (3,4) without (1,3) and with 50 mM mercaptoethanol (2,4); (b) and (c) are the structures of hydrogenated quinolines HQ1 and HQ2.

However, HQ1 having 8-hydroxy-substituent reacts with radicals as a phenol resulting in phenoxyl radical and next quinone formation, whereas HQ2 reacts with radicals as an amine to produce aminyl- and next nitroxyl radicals [51,52]. Figure 2 shows that the mixture HQ1 + RSH demonstrates synergism in oxidative stabilization of methyl linoleate contrary to the antagonistic mixture HQ2+RSH. Synergistic effect can be explained by the ability of thiols to reduce quinones into phenoxyl radicals and phenols and thereby regenerate a strong inhibitor:

$$Q + 2\ RSH \rightarrow QH_2 + RS\text{-}SR.$$

The antagonistic effect of mixtures of aromatic amines (>NH) with thiols in the oxidation of unsaturated methyl linoleate (LH) can be explained by fast reactions of thiyl radicals (RS*) with double bonds, the addition of oxygen to the alkyl radical (RS-LH*) formed, and fast reaction of aminyl radicals with thiols:

$$L'O_2^* + >NH \rightarrow LO_2H + >N^*$$
$$>N^* + RSH \rightarrow >NH + RS^*$$

$RS^* + cis\text{-}LH \Leftrightarrow RS\text{-}LH^* \Leftrightarrow trans\text{-}LH + RS^*$

$RS\text{-}LH^* + O_2 \rightarrow RS\text{-}LHO_2^*$ ($L'O_2^*$)

Water soluble endogenous thiols glutathione and cysteine do not as a rule affect lipid oxidation and efficacy of lipophilic AO. However, in water medium they generate radicals in the reaction with hydrogen peroxide [53]. The yield of radicals is low but it can be enough to initiate thiol-ene reaction with compounds containing double bonds such as resveratrol or caffeic acid [54].

The data presented in this short review may be useful to understanding of physiological role of thiols in the overall oxidative process. It can be expected that further studies of the behavior of thiols in microheterogeneous systems will reveal new reactions and open new opportunities for regulation of red-ox reactions and overcome stressful conditions.

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ВЗАИМНО ВЛИЯНИЕ МЕЖДУ ЛИПИД-АНТИОКСИДАНТ-ПОВЪРХНОСТНО АКТИВНО ВЕЩЕСТВО В МИКРОХЕТЕРОГЕННИ СИСТЕМИ

О.Т. Казайкина

Институт по физикохимия "Н.Н. Семьонов", Руска академия на науките, ул. Косигин 4, 119991 Москва, Руска федерация

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(Резюме)

Този кратък обзор е фокусиран върху съвременното разбиране на ролята на микрохетерогенната реакционна система и по-специално на повърхностно активните вещества (ПАВ) за предотвратяване и инхибиране на окислението на липидите, както и върху влиянието на ПАВ върху отнасянията на познати природни и синтетични антиоксиданти. Дискутирано е и взаимното влияние на компонентите и условията за протичане на синергизъм или антагонизъм в комплексна система (липид-антиоксидант-ПАВ).